

Oral Health in Patients With Type II Diabetes and Impaired Glucose Tolerance

GAIL CHERRY-PEPPERS, DDS, MS
JONATHAN A. SHIP, DMD

OBJECTIVE— To assess the influence of type II diabetes and impaired glucose tolerance on dental, periodontal, and oral mucosal tissues.

RESEARCH DESIGN AND METHODS— We examined 11 subjects with type II diabetes, 32 with impaired glucose tolerance, and 43 control subjects from the oral physiology component of the Baltimore Longitudinal Study of Aging. At the time of the study, none of the participants was taking medication nor being treated for any medical problems other than diabetes.

RESULTS— Only a few statistically significant dental and periodontal changes were apparent in the group with type II diabetes, and no oral mucosal differences existed between the diabetes and control groups. Dental, periodontal, and oral mucosal parameters in patients with impaired glucose tolerance were essentially indistinguishable from the other two groups.

CONCLUSIONS— These findings suggest that among well-controlled individuals with type II diabetes and impaired glucose tolerance, few appreciable differences are evident in oral health.

Type II diabetes and IGT are common chronic metabolic disorders (1) that affect multiple organ systems, including the oral cavity. Good oral health is vital for optimal nutritional intake and deglutition, which is of con-

siderable metabolic relevance for individuals with type II diabetes. Numerous studies have established that periodontal disease is more severe among poorly controlled diabetic patients than control subjects (2,3). Few reported findings are

available on the prevalence of periodontal disease in patients with IGT (3).

Before the development of insulin, uncontrolled type II diabetes was thought to be associated with a marked increase in the incidence of dental caries. Insulin treatment and restriction of sugar intake probably have decreased the incidence of caries. Higher caries rates have been detected in type II diabetic patients with poor metabolic control (2,4). Little information is available on caries and IGT.

Diabetic patients also may have a greater prevalence of oral mucosal lesions than nondiabetic individuals (5). Research has suggested an increased incidence and severity of oral vesiculobulbous conditions and oral lichen planus in patients with type II diabetes (5). The relationship between oral mucosal health and IGT is not well understood.

Evidence also has suggested the level of glycemic control in individuals with altered glucose metabolism plays a strong role in the health of the oral cavity (3). Although previous studies have demonstrated decreased salivary flow rates in diabetic subjects (6), a recent report (7) found no influence of altered glucose metabolism on major salivary gland function in patients with well-controlled type II diabetes or IGT. The purpose of this study was to evaluate the effects of type II diabetes and IGT on dental, periodontal, and oral mucosal health in a well-characterized group of individuals.

RESEARCH DESIGN AND

METHODS— Eleven subjects with type II diabetes, 67.9 ± 11.1 yr of age, with an average disease duration of 7.7 yr; 32 subjects with IGT, 60.7 ± 19.1 yr of age; and 43 control subjects, 60.2 ± 16.8 yr of age were deleted. The data for age are presented as means ± SD. For each patient with IGT and type II diabetes, we selected an age- and gender-matched, nondiabetic control subject.

FROM THE EPIDEMIOLOGY AND ORAL DISEASE PREVENTION PROGRAM, NATIONAL INSTITUTE OF DENTAL RESEARCH, BETHESDA, MARYLAND; AND THE DEPARTMENT OF ORAL MEDICINE, PATHOLOGY, AND SURGERY, UNIVERSITY OF MICHIGAN SCHOOL OF DENTISTRY, ANN ARBOR, MICHIGAN.

ADDRESS CORRESPONDENCE AND REPRINT REQUESTS TO GAIL CHERRY-PEPPERS, DDS, MS, EPIDEMIOLOGY AND ORAL DISEASE PREVENTION PROGRAM, NATIONAL INSTITUTE OF DENTAL RESEARCH, 5333 WESTBARD AVENUE, WESTWOOD BUILDING, ROOM 534, BETHESDA, MD 20892.

RECEIVED FOR PUBLICATION 11 MAY 1992 AND ACCEPTED IN REVISED FORM 12 NOVEMBER 1992.

TYPE II DIABETES, NON-INSULIN-DEPENDENT DIABETES MELLITUS; TYPE I DIABETES, INSULIN-DEPENDENT DIABETES MELLITUS; IGT, IMPAIRED GLUCOSE TOLERANCE; OGTT, ORAL GLUCOSE TOLERANCE TEST; WHO, WORLD HEALTH ORGANIZATION; ANOVA, ANALYSIS OF VARIANCE.

Table 1—Dental parameters

	DIABETIC SUBJECTS	IGT SUBJECTS	CONTROL SUBJECTS
N	11	32	43
TEETH (N)	24.3 ± 4.5	21.8 ± 7.9	27.0 ± 5.7
THIRD MOLARS (N)	0.1 ± 0.3	0.9 ± 1.6	0.7 ± 1.3
CORONAL CARIES SURFACES (N)	3.8 ± 9.5 ^{a,b}	0.6 ± 1.2 ^{*b}	0.7 ± 1.7 ^{*a}
CERVICAL CARIES SURFACES (N)	1.0 ± 1.2	0.2 ± 0.5	1.3 ± 4.8
CORONAL RESTORATION SURFACES (N)	30.0 ± 13.0	38.9 ± 29.6	37.0 ± 21.6
CERVICAL RESTORATIONS SURFACES (N)	3.6 ± 4.5	2.0 ± 2.7	2.3 ± 3.3
MISSING SURFACES (N)	20.3 ± 19.6 ^{*c}	29.0 ± 36.7 ^{*c}	16.9 ± 26.8
DECAYED, MISSING, FILLED TOOTH SURFACES (N)	53.8 ± 29.7	70.7 ± 35.9	56.9 ± 33.9

Data are means ± SD.

*Columns that share a common subscript are significantly different ($P < 0.01$).

All subjects were volunteer participants in the oral physiology component of the Baltimore Longitudinal Study of Aging (8), conducted by the National Institute on Aging. The subjects were healthy whites of middle socioeconomic class, who visited the dentist at least once per year. At the time of the study none was taking any medications or being treated for medical problems other than type II diabetes. All type II diabetic subjects used diet to control their disorder. Each participant underwent extensive laboratory and medical examinations to assess their health status (8).

An OGTT was performed in the early morning after an overnight 10- to 16-h fast (9). Each participant received an oral glucose load of 40 g/m² body surface area. Sampling was done at 20-min intervals for 2 h, within 3 yr of their oral examination. Participants were diagnosed with 2-h glucose values, according to WHO criteria (10). All participants with normal glucose values were considered control subjects. Glucose tolerance tests were not routinely performed on all diabetic subjects (control subjects = 114 ± 2.3 mg/dl; IGT = 159 ± 8.2 mg/dl, mean ± SE). Status of subjects with type II diabetes was assessed with the Bio-Rad Micro Column Test (Richmond, CA) (11) to measure serum HbA_{1c} levels (HbA_{1c} = 6.9 ± 3.1%, mean ± SE).

One examiner performed the oral examinations (J.A.S.). All dental, gingival, and periodontal parameters were assessed according to criteria established by the National Institute of Dental Research (12). The mesiobuccal and mid-buccal surfaces of all teeth were probed, and pocket depth, recession, and attachment loss measurements computed. An index (13) was used to establish the extent and severity of periodontal attachment loss. Two standardized extra- and

intraoral mucosal examinations assessed the clinical appearance of the oral mucosa (14,15).

Statistical analysis

Age- and gender-matched control groups were formed separately for the type II diabetes and IGT groups. No statistical differences were detected between the two control groups for any of the oral health parameters, and therefore, one control group was used. ANOVA tests determined dental, gingival, and periodontal parameters for the three groups. When an ANOVA test was statistically significant, further analyses were conducted. The frequency of oral mucosal diagnoses among the groups was compared with two-sided Fisher's exact tests and χ^2 tests ($P < 0.01$).

RESULTS— Most dental parameters were not statistically different among the three groups (Table 1). The type II diabetes group had more surfaces with coronal caries than the others, and the IGT group had more missing teeth than the type II diabetes group. The gingival findings indicated that gingival health

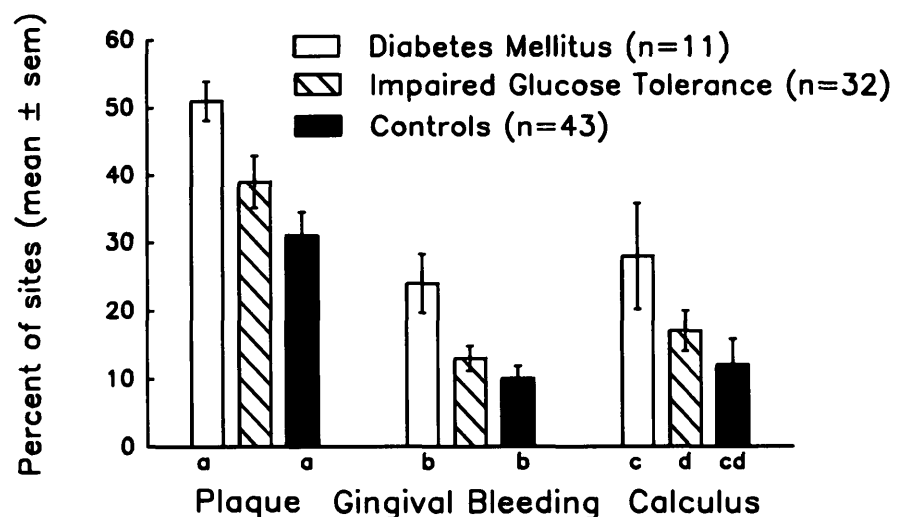


Figure 1—Gingival parameters in subjects with type II diabetes, IGT, and control subjects. Columns that share a common subscript are significantly different ($P < 0.01$). Data are means ± SE.

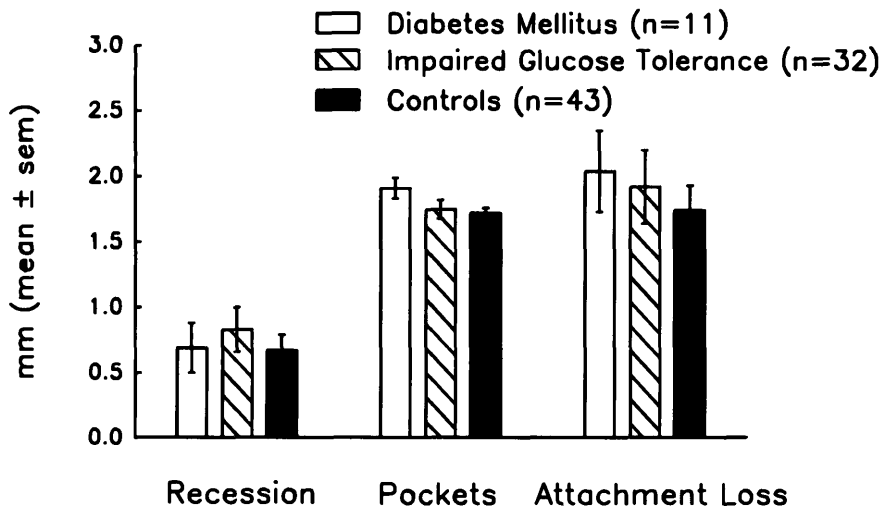


Figure 2—Periodontal parameters in subjects with type II diabetes, patients with IGT, and control subjects. Data are means \pm SE.

was poorer among the diabetic subjects (Fig. 1).

The type II diabetes group had a greater prevalence of sites with dental plaque, gingival bleeding, and calculus compared with the control and IGT groups. The IGT group also had an increased percentage of sites with calculus compared with the control group.

No statistical differences among the three groups were found for periodontal measurements (Fig. 2), nor in the Extent and Severity Index (13). Oral mucosal findings using two diagnostic criteria (14,15) revealed no statistical differences among the three groups ($P > 0.01$). The most frequent diagnosis in all three groups was normal oral mucosa.

CONCLUSIONS— These results demonstrate minimal oral changes in subjects with type II diabetes, and essentially no changes in those with IGT. The type II diabetic population was in good health, received regular medical and dental care, and was well controlled (average HbA_{1c} = 6.9%, duration 7.7 yr). All participants were well-characterized, unmedicated, and being treated for no other medical problems. The sample size of the type II diabetes group

was small; therefore, these findings may not be applicable to other populations.

In general, the three groups had few differences in dental caries. The diabetes group had more surfaces with coronal caries, which may have occurred when they were in a poorer glycemic state. Previous research documents that the incidence of caries is higher in poorly controlled diabetic patients, whereas well-controlled diabetic patients and nondiabetic control subjects showed no differences (4). Diabetic patients with diminishing metabolic control may be more susceptible to dental caries than well-controlled type II diabetic subjects and nondiabetic subjects (2,4).

Although all three groups in this study displayed good oral hygiene, adult type II diabetic subjects experienced an increase in gingivitis (Fig. 1), which is consistent with previous findings (3). Diabetic patients are susceptible to infections by bacteria that colonize the gingival sulcus and result in increased plaque, inflammatory changes in the gingiva, and possible periodontal destruction (3). None of the groups in our study exhibited differences in periodontal measurements, however (Fig. 2). Gingivitis may not have been present for a sufficient

period of time to progress to periodontitis. In addition, the level of diabetic metabolic control may determine susceptibility to periodontal disease.

The prevalence of oral mucosal lesions was low in subjects with type II diabetes or IGT. Previous reports have found increased lesions in diabetic patients (5), but their level of glycemic control is unclear. Therefore, glucose tolerance per se seems to have no effect on the clinical appearance of the oral mucosa in subjects with well-controlled type II diabetes or IGT.

In this study, the oral health of subjects with IGT was similar to that of control subjects. IGT is a less complex metabolic disease than type II diabetes and typically has fewer oral complications (16). Frequent use of dental and medical services, good glycemic control, and a moderate duration of disease history (7.7 yr) most likely account for the higher level of oral health in the study population compared with other studies. Future analyses of type I and II diabetes with varied glycemic states may confirm the associations of oral disease and type I and type II diabetes.

Acknowledgments— The authors greatly appreciate the assistance of Dr. Reubin Andres, Dr. John Sorkin, and other staff in the Metabolism Section, National Institute on Aging, Baltimore, Maryland. We also thank Dr. Bruce Baum for his guidance and support, and Dr. Ingrid Valdez for her critical review of the manuscript.

References

1. National Diabetes Data Group: Classification and diagnosis of diabetes mellitus and other categories of glucose intolerance. *Diabetes* 28:1039–57, 1979
2. Galea H, Aganovic I, Aganovic M: The dental caries and periodontal disease experience of patients with early onset insulin dependent diabetes. *Int Dent J* 36: 219–24, 1986
3. Sastrowijoto SH, van der Velden U, van

- Steenbergen TJM, Hillemans P, Hart AAM, de Graaff J, Inpijn L: Improved metabolic control, clinical periodontal status, and subgingival microbiology in insulin-dependent diabetes. *J Clin Periodontol* 17:233–42, 1990
4. Pohjamo L, Knuutila M, Tervonen T, Haukipuro K: Caries prevalence related to the control of diabetes. *Proc Finn Dent Soc* 84:247–52, 1988
 5. Grinspan D, Diaz J, Villapol L, Schneiderman J, Berdichefsky R, Palese D, Faerman J: Lichen ruber planus de la muqueuse buccale. Son association a un diabete. *Bull Soc Francaise* 73:898–99, 1966
 6. Conner S, Iranfour B, Mills J: Alteration in parotid salivary flow in diabetes mellitus. *Oral Surg* 30:55–9, 1970
 7. Cherry-Peppers G, Sorkin J, Andres R, Baum BJ, Ship JA: Salivary gland function and glucose metabolism. *J Gerontology* 47:M130–34, 1992
 8. Shock NW, Greulich RC, Andres R: Normal human aging: the Baltimore Longitudinal Study of Aging. Washington, DC, U.S. Govt. Printing Office, (NIH publ. no. 84–2450)
 9. Shimokata J, Greulich R, Muller D, Fleg J, Sorkin J, Ziemba A, Andres R: Age as an independent determinant of glucose tolerance. *Diabetes* 40:44–51, 1991
 10. World Health Organization: *WHO Expert Committee on Diabetes Mellitus: Second Report*. Geneva, World Health Org. 1980 (Tech. Rep. Ser. no. 646)
 11. Biorad: Hemoglobin A1c Micro Column Test: Manual no. 192-8000, Aug 1987
 12. Miller AJ, Brunelle JA, Carlos JP, Brown LJ, Loe H: *The National Survey of Oral Health of United States Adults: 1985–1986*. National Institutes of Health, Public Health Service, Department of Health and Human Services, Washington, DC, U.S. Govt. Printing Office, 1987 (NIH publ. no. 87–2868)
 13. Carlos JP, Wolfe MD, Kingman A: The extent and severity index: a simple method for use in epidemiologic studies of periodontal disease. *J Clin Periodontol* 13:500–505, 1986
 14. National Health and Nutrition Examination Survey III, Oral Health Survey Methods. Rockville, MD, Westat, 1984
 15. Wolff A, Ship J, Tylenda C, Fox P, Baum B: Oral mucosal appearance is unchanged in healthy, different-aged persons. *Oral Surg Oral Med Oral Path* 71: 569–72, 1991
 16. Sastrowijoto SH, Abbas F: The relationship between bleeding/plaque ratio and family history of diabetes mellitus and impaired glucose tolerance. *J Clin Periodontol* 17:55–60, 1990